



## FENS Forum 2010 - Amsterdam

- Posters: to be on display from 8:00 to 13:15 in the morning and from 13:30 to 18:45 in the afternoon. Poster sessions run from 09:30 to 13:15 in the morning and from 13:30 to 17:30 in the afternoon. A one hour time block is dedicated to discussion with the authors (authors should be in attendance at their posters as from the time indicated.)
- For other sessions, time indicates the beginning and end of the sessions.

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**First author** Min, Rogier (poster)

Poster board B39 - Mon 05/07/2010, 14:30 - Hall 1

Session 103 - Synaptic plasticity 3

Abstract n° 103.39

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**Title** Mechanisms underlying the induction and expression of spike-timing dependent depression at L4-L2/3 synapses in barrel cortex

**Text** Excitatory synapses in the neocortex are able to undergo bidirectional modifications of synaptic strength depending on the relative timing of pre- and postsynaptic activity (spike timing-dependent plasticity; STDP). STDP in primary sensory cortex is essential for the proper formation of sensory circuits. However, the signaling cascades underlying these changes in synaptic strength are still not completely unraveled. Postsynaptic calcium influx is thought to be of primary importance in determining the direction and magnitude of STDP, but research from our group has shown that the postsynaptic calcium signal by itself is not enough to predict the direction of STDP. Indeed, it has been shown that the signaling cascades leading to spike timing-dependent potentiation and spike timing-dependent depression show important differences.

Here, we studied STDP at synapses between L4 spiny stellate and L2/3 pyramidal neurons in rat barrel cortex. Both spike timing-dependent potentiation and spike-timing dependent depression at this synapse require activation of glutamate receptors of the NMDA receptor (NMDAR) family. But while spike timing-dependent potentiation requires the activation of postsynaptic NMDARs, spike timing-dependent depression involves activation of presynaptic NMDARs. Furthermore, depression also requires endocannabinoid signaling, presumably through presynaptic CB1 receptor activation. It is currently unclear how presynaptic NMDAR activation and endocannabinoid signaling act in concert to achieve synaptic depression. We use two-photon fluorescence Ca<sup>2+</sup> imaging of both the pre- and the postsynaptic compartment of L4 to L2/3 connections to study the pathway leading to this reduction in synaptic efficacy. In this way, we try to identify the signaling cascades involved in the induction and expression of spike timing-dependent depression.

**Theme** B - Excitability, synaptic transmission, network functions  
Synaptic plasticity - Spike-timing dependent plasticity

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